intraperitoneal fluid, and marked splenic enlargement. Computed tomography confirmed the cystic abnormality in the left lobe to be in continuity with the left main hepatic duct. The appearance of the liver suggested macronodular cirrhosis with associated portal hypertension. Hydatid serology was negative and serum tumour markers (α-fetoprotein, CA 19–9) were normal. His liver function tests had worsened despite there being no clinical evidence of ongoing sepsis (table 1).

Questions

(1) What does the PTC (fig 1) show and given the history of ulcerative colitis what is the most likely diagnosis?
(2) What is the connection between this hepatological diagnosis and the findings of dysplasia at colonoscopy?
(3) Given the deteriorating liver function tests, computed tomography findings, and colonoscopic changes how would you manage this case?

An elderly man with pleural effusion and abnormal behaviour

A S Kashyap, S Kashyap

Answers on p 413.

A 76 year old man, who had been an asbestos insulation worker, presented with a four month history of right sided severe diffuse chest pain, breathlessness on exertion, and weight loss of 10 kg. He had recurrent episodes of lethargy, agitation, and abnormal behaviour. These episodes were partially relieved by eating a high carbohydrate diet. On admission he was found to be unrousable. His Glasgow coma scale score was 7/15 with no focal neurological deficit. He had clubbing and right sided massive pleural effusion and contracted right hemithorax with mediastinal shift to same side. Full blood counts, urea, electrolytes, liver function tests, and serum calcium were normal. Plasma glucose concentrations were 1.5 mmol/l. He was given 50 ml of 50% dextrose intravenous with correction of his hypoglycaemia and he became conscious. A chest radiograph (posteroanterior) showed complete opacification of right hemithorax (fig 1). Computer tomography of his thorax revealed right pleural thickening encasing the collapsed right lung and contraction of right hemithorax. Pleural aspiration revealed a viscous jelly-like exudate with "atypical" mesothelial cells and high hyaluronidase levels. Pleural biopsy revealed a malignant sarcomatoid mesothelioma. The subsequent week was characterised by rapid reaccumulation of pleural effusion in spite of repeated aspirations. The hypoglycaemic episodes increased in frequency though he was on approximately 380 g of glucose intravenously per day with intramuscular glucagon intermittently.

Plasma glucose concentrations were 0.8 mmol/l, insulin (<25 pmol/l), C peptide (<75 pmol/l), and β-hydroxybutyrate (<20 mmol/l) were undetectable, serum growth hormone was 0.9 mU/l. Insulin-like growth factor-II (IGF-I) concentration was 0.16 U/ml (reference range 0.4–2.0), serum IGF-II concentration was raised at 2.0 U/ml, and ratio of IGF-I: IGF-II was 0.08 (normal >0.2). "Big" IGF-II concentration was 20.8 nmol/l (0–14.4) and IGF binding protein-3 (IGFBP-3) concentrations were 2.2 mg/l (2.0–4.8). A short tetracosactrin (Synacthen) test was normal.

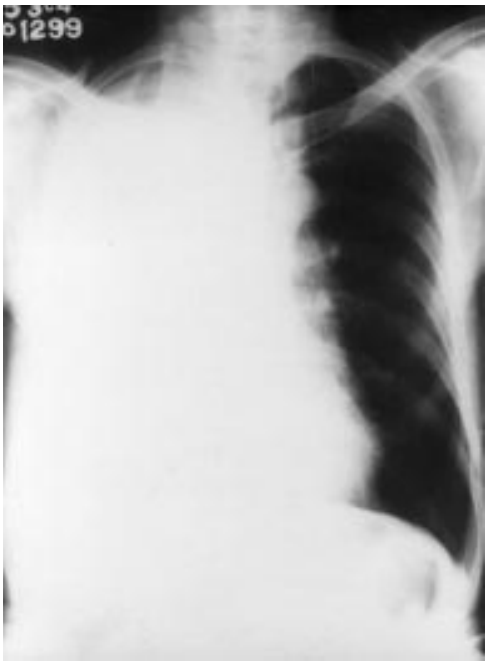

Figure 1 Chest radiograph (posteroanterior) showing complete opacification of right hemithorax.

Questions

(1) What is the cause of this patient's hypoglycaemia?
(2) What pharmacological agents may alleviate his hypoglycaemia?
(3) What is the role of surgery in management of this patient?

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A difficult case of gastrointestinal haemorrhage

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Answers on p 414.

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A 64 year old man presented to the accident and emergency department, having been found collapsed in the street. He gave a four day history of melaena. On examination he was shocked, with a pulse of 140 beats/min and a blood pressure of 70/40 mm Hg. Initial haematological assessment revealed a haemoglobin concentration of 65 g/l, platelet count 68×10^9/l, and international normalised ratio 1.3. After resuscitation with intravenous colloid fluids, blood, fresh frozen plasma and platelets, an emergency upper gastrointestinal endoscopy was performed. This showed four large oesophageal varices, two of which were actively bleeding. Endoscopic sclerotherapy was attempted, but haemostasis was not achieved, and a Sengstaken tube was sited.

The next day the Sengstaken tube was removed and endoscopic band ligation of the oesophageal varices performed. The patient remained stable for 24 hours until there was evidence of further gastrointestinal bleeding, with passage of melaena, and haemodynamic compromise. A further upper gastrointestinal endoscopy was performed, which revealed a large quantity of blood in the stomach; the oesophagus was well visualised and confirmed not to be bleeding. A therapeutic procedure was performed (fig 1).

The procedure successfully stopped further gastrointestinal haemorrhage. However, two days later, he became drowsy and disorientated.

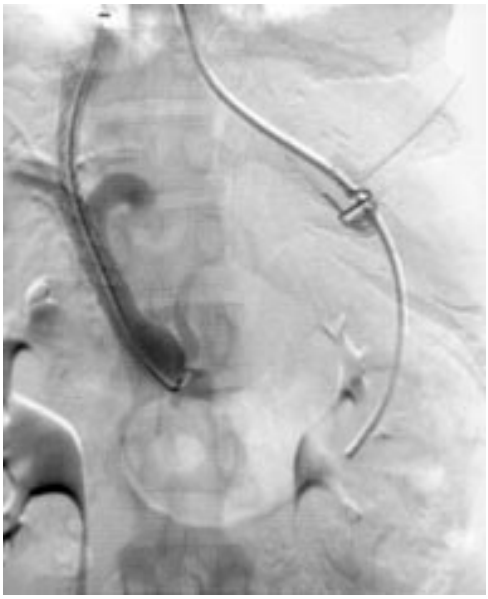

Figure 1 Therapeutic procedure performed on patient.

Questions
(1) Following the insertion of the Sengstaken tube what additional management would you have instigated?
(2) What was the cause of the rebleed and what procedure was performed?
(3) What is the likely cause for the patient becoming drowsy, and how would you manage this?

Recurrent pulmonary oedema in a 53 year old woman

S G Williams, S J Lindsay, L B Tan

Answers on p 416.

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A 53 year old woman presented with pulmonary oedema after an inferior myocardial infarction three months earlier. Blood pressure on admission was 210/120 mm Hg. Initial treatment included diuretics, nitrates, and oxygen and her symptoms settled. Echocardiography before discharge showed mild impairment of left ventricular contraction. Coronary angiography performed several months later showed an occluded right coronary artery, with a 50% stenosis of the left anterior descending vessel. Overall left ventricular function was good (ejection fraction of 58%) with some regional inferior hypokinesia. Over the next two years, the patient was admitted on numerous occasions with hypertensive pulmonary oedema and was treated medically. Between admissions she remained symptom free although blood pressure control was difficult.

Questions
(1) What is the most likely cause of this patient's recurrent pulmonary oedema?
(2) What investigations should be performed next?
(3) What treatment options are available to this patient?